

INTRODUCTION

Parasites, diversity and the ecosystem.

Peter Hudson¹

1. Center for Infectious Disease Dynamics, Biology Department, Penn State University,
University Park, Pa 16802

1.1 The dualities of parasitism

Dualism is a dominant theory of life that considers reality to be a balance between two independent and fundamental principles: good and evil, mind and matter, nature and nurture. In the same manner we see the thread of dualism run through the ecology of parasitism: they can generate diversity but cause extinction, they may castrate a host but increase its growth rate, they can stimulate an immune response but at the same time encourage a secondary chronic infection. Parasites inhabit individual hosts that are distributed as discrete patches, much like a metapopulation but these hosts are also nested within a spatially structured metapopulation and these within a meta-community of competent hosts. They often divert the host's resources to themselves and away from other consumers and so change energy flow patterns, the use of critical resources and so influence ecosystem functioning. The majority of living organisms are parasitic and their role as specialist consumers and their influence on biodiversity may well make them important players in many ecosystems.

Given the rather special and probably pivotal role parasites may play in many ecosystems, it is somewhat surprising that few workers have considered the role of parasitism at the ecosystem level. Probably the central question is to ask, how do parasites influence ecosystem functioning? Or more specifically, what are the consequences of parasite removal for the community and energy flow in the ecosystem? What is the biomass of parasites within the ecosystem and how does this compare with other natural enemies? How do the parasites influence the flow of specific chemicals and minerals through the system? How do parasites influence biodiversity? And how does biodiversity influence parasitism? Questions that we are only just starting to get vague answers to but nevertheless the questions that are the underlying driving force behind the production of this book. There is a common assumption that parasite biomass is negligible (e.g. Polis 1999) but is this assumption really correct? To illustrate the sheer significance of parasites in an ecosystem let me tell you about a comment made by my friend and colleague, Armand Kuris. He once asked me what I thought was the biomass

of parasites on the Carpinteria saltmarsh (about 70 ha) where he and Kevin Lafferty have studied the trematodes of the gastropod and bird fauna for many years. I had not a clue, looked deep into my glass of wine and fumbled with kilograms. "Our provisional estimate is in the order of several elephants (if they weigh 3 tons maybe as many as 7-10) with a reproductive rate equivalent of several babies per year (maybe as high 1-2 baby elephants per day) for the 200 warmest days of the year" he replied. Astonishing, absolutely amazing and as a card carrying parasitologist, I was embarrassed by my lack of comprehension. Just imagine a small herd of elephants on a wetland in Southern California, they would be considered a dominant feature and if they were just consuming vegetation they would have a fantastic impact on the environment, especially in that small area of habitat. But these parasites are living off snails and birds and that high reproductive rate they must be having a huge impact on the growth rate of their hosts, influencing the flux of energy to other trophic level and shaping community structure by reducing competitive abilities of their hosts and vulnerability to predation. Of course this is only part of the potential impact of the parasites on the ecosystem since Kuris and Lafferty have not estimated the biomass of the plant pathogens, many of the parasites in the crustacean or in the tertiary consumers

Asking good questions and making estimates of parasite biomass can help us to get the role of parasites in perspective but the answers to the questions are far from easy and we should appreciate the amount of hard work that has gone behind the studies of parasites in the Carpinteria salt marshes of Californian. To make some of the questions answerable, we may need to restructure them into a form that can be answered, perhaps by starting at the level of the individual and then using this foundation of understanding to explore issues at population, community and ecosystem level. Hence we may ask, if parasites have an impact on the individual host and what are the emergent properties we may observe at the population and community level? How does the parasite interact with other natural enemies and then what are the consequences of these interactions to ecosystem functioning? This approach is based on undertaking insightful experiments at the lower level, monitoring changes in the intensity of parasites and age related effects and then integrating our understanding through models and identifying the patterns and

emergent features we would predict to observe at the higher levels. Another approach is to examine an ecosystem that is subject to an epidemic. For example, what happened to the marine ecosystem of the North sea when Phocine distemper virus reduced the population of seals? Did fish survival change? Did seabirds compensate with improved breeding production and survival or were such events so transitory as to have little influence? Again modeling and understanding can provide insights. Another dominant approach to examining ecosystems effects is to apply the comparative method to identify patterns and then dissect the data to propose the putative mechanisms. The chapters in this book use all of these techniques and together provide an integrated and clear examination of parasites at the ecosystem level.

This short chapter serves as an introduction. I shall try to lay the scene for the role of parasites in ecosystems and in doing this I have to admit I face the tensions of my own inner duality. On the one hand I find the task daunting, our knowledge vague and the scale of the issue massive and I am aware that focusing on the parasite component of an ecosystem may inadvertently trivialize other critical components. On the other hand, the task is exciting and a challenge that we should rise to: pathogens and parasites have not been included in the theories of trophic structure and are frequently ignored from ecosystem ecology (Polis & Strong 1996, Polis 1999), we have growing understanding of the role of parasites, some excellent modeling approaches and the time is ripe for a book like this. So to set the scene for the book I will look at a specialist parasite and a generalist parasite in two contrasting foodwebs to examine how they influence community structure and the ecosystem. I shall pick some fundamental questions that examine the role of parasites in ecosystems and illustrate those with a few examples, selecting some examples that are not used by others in the book and of course shamelessly referring to my own work.

1.2 Specialist and generalist parasites in the ecosystem

While the study of parasites was once the sole domain of the specialist parasitologist, often focused on the difficult and challenging task of working out the life cycle and taxonomic position of parasites, it is now apparent that this fundamental biological knowledge has allowed parasitology to come of age so that a wide range of scientists and disciplines now addressing parasitological questions. The issues scale from the molecular to the ecosystem and to my mind, the challenge for the future is to ensure that the discipline becomes integrated vertically so that an understanding of the processes of infection and persistence at the molecular level can be incorporated in making predictions about the temporal and spatial spread of diseases and in identifying how parasitism influences ecosystem functioning. For example, parasites often weaken their hosts, making them morbid, and thus susceptible to predation (Hudson et al 1992b, Packer et al 2004). How does this influence the way energy flows through the foodweb? the consequences on the demographics of the host, the predator, the parasite and the competitors in the system?

Understanding the importance of species and groups within an ecosystem is one of the central challenges to ecology and so if we are to investigate the role of parasites, the design of the question is important. Probably one of the most important questions would be to ask: if we were to remove the parasite from the system what would be the consequences? You may predict that host population growth rates would rise and would then lead to an increase in the growth rate of the other consumers of that host but then maybe they would suppress our host through the classic process of the paradox of enrichment. Alternatively, you may predict that the parasites keep the prey unhealthy allowing the predators to catch and obtain a feed so the removal of parasites makes the prey healthy and the predators starve so the host population would rise to be regulated by some other factor such as food availability. On the other hand some other parasite may invade the niche and perhaps one that used the predator as an obligate host and then changed the dynamics of the whole community. So this apparently simple question is not trivial but we can quite quickly see there are a suite of mutually exclusive hypotheses to test in the wild. I suspect the true answer to the role of the parasite depends on how the parasite-host relationship is embedded in the food web and more specifically whether the

parasite is a generalist shared between species or a specialist. In a simple food web when we have a single host infection that shapes the population dynamics of the host then this can be dominating and have far reaching repercussions to the whole community. I will try to illustrate by studies on red grouse and their caecal nematode, *Trichostrongylus tenuis*. If grouse lived in a more complex ecosystem then the effects of this specialist parasite may well be buffered by the other interactions. Much to my dismay, grouse do not live in the Serengeti, one of the more fascinating ecosystems in the world and one with a relatively complex food web. I know of no detailed studies on a specialist parasite in this system but there have been studies on a number of interesting generalist parasites. Rinderpest infected many of the ungulate species and appeared to have led to a dramatic and far reaching change in the ecosystem, so we shall examine this system as a contrast to the grouse system.

1. 2.1 **Rinderpest in African ungulates:**

Rinderpest is a disease of ungulates caused by a morbillivirus and an equivalent of a “buffalo measles” transmitted through an aerosol of virus during coughing and sneezing. This is an infection of domestic animals that invaded Africa in about 1889 and then spread at a frightening pace across the continent to reach the Cape within just 8 years (more details in Chapter 8). The impact on wild ungulate populations was dramatic, Buffalo and Wildebeest were decimated by about 95% and local populations of greater kudu, bongo and eland were totally wiped out. Here was a huge “experimental” perturbation to the ecosystem of the African savannah that is probably still influencing the functioning of several ecosystems today, more than 100 years later.

We probably know most about the situation in the Serengeti than elsewhere since the disease became endemic in the park (Dobson 1995). In the 1960s there was a heavy vaccination program that eventually ringed the whole park and by 1968 the disease was eradicated. At that time there was a series of detailed and fascinating scientific studies that have since followed changes in the population of ungulates and the ecology of the area and so recorded the recovery of the ungulates and changes in the ecosystem since rinderpest. Over this period, the wildebeest and buffalo exhibited what the Serengeti

workers describe as “an eruption”: wildebeest and buffalo increased dramatically, at a rate of about 10% per annum, so wildebeest numbers were up seven fold in 17 years and buffalo showed a parallel increase. There is a circumstantial evidence that this increase was a consequence of rinderpest since other grazing species like the zebra (not an ungulate) were unaffected. Furthermore, subsequent epidemics all provide good evidence that Rinderpest was a significant factor limiting the size of several ungulate species. The interesting feature is the effect this must have had on the rest of the ecosystem. Sinclair (1979) examined the effect that the eruption of wildebeest had on the Serengeti ecosystem; the increase in the wildebeest changed the seasonal grazing pattern and the abundance of the grasses and the herbs, reduced the combustible material and reduced fires that in turn allowed tree regeneration and through these processes influenced the whole community of herbivores. The wildebeest compete with the buffalo so while buffalo increased after the removal of rinderpest, the population subsequently leveled off, probably because of competition for food from the wildebeest. They probably influenced the other grazers such as zebra (-ve: direct competition) Grants gazelle (+ve: herbs increased) and giraffe (+ve: trees increased). While there was some evidence that the predator populations responded to the increased prey base (Spinage 1962) the evidence is not clear, probably because wildebeest are seasonal migrants and the predator populations are regulated by the prey base during the intervening periods but also because a subsequent increase in predators led to the outbreak of other diseases (see Dobson 1995 and Chapter 8).

While these data are limited and the story is pieced together from anecdotal evidence, Sinclair’s deep understanding of the natural history of the Serengeti and Dobson’s analysis, it illustrates well how the removal of a pathogen can shape the processes within an ecosystem. Essentially the pathogen acted to reduce the abundance of the primary consumers and so influenced competition with other grazers, the vegetation structure and no doubt the flow of energy through the ecosystem.

1.2.2 Parasitic worms in British grouse:

Red grouse inhabit the open, semi-natural moorlands characteristic of the British uplands where the vegetation is dominated by heather (*Calluna vulgaris*), the primary food plant of the red grouse. While the red grouse is the only species that relies solely on the heather, the habitat is home to a number of other species including the mountain hare, red deer, roe deer and an avifauna that is predominantly migratory. These large tracts of heather moorland are managed by keepers to produce a harvestable surplus of grouse each year and provide grazing for the sheep farmers. The grouse are preyed on by foxes, golden eagles, hen harriers, short eared owls and peregrines but the keepers legally control the foxes and frequently interfere or kill the protected raptors (Thirgood et al. 199x).

The grouse exhibit unstable population dynamics with oscillations in abundance and a period of usually between 5 and 12 years. The maximum growth rate of the grouse is determined by the quality of the main food plant but subsequent changes in abundance are a tension between the natural enemies of the grouse (Hudson et al 2002). Grouse are infected with a caecal nematode which reduces their condition and breeding production and these demographic effects coupled with the low degree of parasite aggregation in the host population generates instability in the population that can account for the cyclic fluctuations in red grouse abundance recorded in harvesting records (Hudson et al 1992a, Dobson & Hudson 1992). Large scale experiments that have removed parasites at the population level effectively stop the periodic crashes in abundance indicating that parasites play an important role in the cyclic nature of this species (Hudson et al 1998). So while the direct interaction between parasite and host plays a major role in generating the cyclic fluctuations in abundance, we should now ask how this parasite-host interaction shapes the effects of other natural enemies in the ecosystem.

The parasites major influence on the host is to reduce body condition and make the host morbid, thus less able to produce young (Hudson 1986), defend territories (Fox & Hudson 1992) and more vulnerable to predation (Hudson et al 1992b). The grouse emit a characteristic scent which trained pointing dogs can smell at remarkable distances, when the hens commence incubation they close off their caeca and no longer produce caecal

faeces and at the same time stop emitting scent the trained dogs can locate. It is here, in the caeca, that the parasitic worm lives and interferes with the workings of the caeca (Watson et al 1985). Highly infected grouse have difficulties controlling their scent emission and the dogs, searching by scent, can locate these grouse significantly more frequently than individual who have had their worms experimentally removed (Hudson et al 1992b). Incorporating this selective predation into a model of parasite-host interaction predicts an increase in the host population (Hudson et al 1992b, Packer et al 2004); initially a counter intuitive finding since we would not expect the addition of predation mortality to increase prey abundance (Figure 1). However, since the predation is selective, the predator is removing the heavily infected individuals from the population, thus reducing the regulatory role of the parasite, dampening the oscillatory behaviour of the population and leading to an overall increase in the population. Interestingly, one may predict that harvesting by humans, another form of predation, and should also remove parasites and lead to dampening of the cycles but an examination of the time series data shows this is clearly not the case (Hudson & Dobson 2000). In fact what seems to be happening is that much of the infection process has already taken place prior to the start of harvesting. The infective stages are on the ground before the harvesting commences so the “dye is cast” and the infection process will continue irrespective of any removal of grouse by the hunters although reduced density over an extended period will of course lead to reduced infection levels.

Grouse are also infected with a tick borne virus that causes the disease Louping ill, a significant cause of mortality in populations where it is prevalent. The ticks themselves cause little mortality unless numbers are high but in areas like the North Yorkshire Moors the grouse pick up the ticks from bracken dominated ground, a habitat that provides high humidity and assists tick survival (Hudson 198x). In the past, bracken was cut for livestock bedding and these activities restricted the bracken beds to the steep slopes but poor heather burning practices coupled with heavy grazing has allowed the bracken to escape from these slopes and invade the heather moorland thus bringing the ticks into the habitat used by the grouse and exposing them to infection. The tick life cycle is dependent on the presence of a large mammalian host and the removal of these hosts

should lead to the eradication of the tick and the louping ill. In areas where the sole host is the sheep then sheep can be treated with acaricides and the ticks eradicated. In other areas, deer may be important hosts for tick but (unlike the sheep) they are not competent hosts for the virus, thus deer act as a “sink” for virus but a “source” for ticks (Gilbert et al 2000). In effect, the deer act as “dilution hosts” for the virus. Theoretically, if the deer host a large proportion of the ticks then they can reach a point where more virus is lost through these “wasted bites” into the dead-end deer host than is generated from the competent hosts the virus levels fall. However there is another interesting player in this ecosystem: the mountain hare. Experimental studies have shown that while mountain hares do not permit direct viral amplification through the normal systemic route (like deer) they do permit non-systemic transmission between co-feeding ticks, unlike the deer (Jones et al 19). Laurenson et al (2003) undertook a large scale experiment where they removed hares from a large area of moorland habitat and showed a significant decrease in the louping ill seroprevalence in the grouse. Interestingly they were able to show that much of this decline in infection was because they removed the effects of co-feeding transmission. In other words, the hares were the key player that kept the virus persistent in this ecosystem and while the hares were a source of ticks the important role they played was in providing a suitable habitat that permitted transmission between ticks. The final outcome of the role played by the virus is in the interplay between hares, deer and grouse as hosts for both ticks and louping ill. The whole vertebrate community plays a role in determining louping ill dynamics but the specialist grouse-nematode interaction has far reaching repercussions to the predators and the balance between these mortality factors and then the quality of the vegetation molds the grouse dynamics.

Both of these studies are interesting since they illustrate the role of parasitism in the ecosystem. An understanding of the simple parasite-host relationship at the individual level allowed an understanding of population dynamics, the interaction with other natural enemies and how changes in the community structure influenced the ecology within the ecosystem. The Serengeti is a unique and rich ecosystem of international significance that rightly provides a wildlife spectacular but is vulnerable to invasive diseases. We just mentioned rinderpest but we could also have a range of other infections such as rabies,

canine distemper virus, anthrax and bovine tuberculosis and the ways these pathogens influence ecosystem functioning. Dobson (1995) has suggested that these have changed in prevalence as a consequence of changes in the ungulate population. This interplay between species composition, abundance and disease prevalence is found in both the Serengeti and the Heather moorlands of Britain and together probably play an important role in shaping community structure and ecosystem functioning. The North American equivalent is Yellowstone with Bison, elk, wolves and bears. Here the Bison population has increased dramatically following the grooming of roads after snow fall and so brucellosis has become a major concern, not directly to the Bison population but indirectly by the perceived threat wildlife pose as reservoirs of infection to cattle on neighboring ranches. In the same vane, humans also interact with the semi-natural heather moorland where grouse management is a dominant form of land use in the uplands of Britain. The grouse management favors a multiple land use system that incorporates sheep farming, conservation and recreation but if the economics of this collapse then this land is sold for commercial forestry, a single land use system of little benefit to wildlife. But there is an irony here, another one of the dualities of parasitism if you like. The significance of the disease to the grouse has arisen primarily because the keepers reduce the predation pressure from the foxes so allowing grouse numbers to rise and parasitism to become a problem. This parasitism leads to highly unstable dynamics so that every few years the disease forces the grouse down to low levels. At this point if the predators are allowed back then the grouse will be held at this low level, the grouse no longer become a viable crop to harvest and the land can be sold to a single land use such as commercial forestry. The parasites are quite capable of knocking the host population down to a low density that inverse or simple density dependent effects prevent them from rising.

These two examples show us that a specialist parasite like the nematode worm in the grouse can have important implications for the population dynamics of the host and consequently other natural enemies. For a generalist pathogen like rinderpest or even Louping ill (a specialist pathogen but with a generalist vector) has important

repercussions to the whole community. We have evidence to suppose parasites should be important to the ecosystem.

1.3 Does biodiversity affect parasitism?

There is increasing evidence that species composition and diversity influence ecosystem functioning (Tillman 1997) and productivity (Tillman et al 2001). These studies and others are showing us that biodiversity really matters to the quality of our environment and since we are loosing biodiversity fast we are changing ecosystem functioning. But what are the processes involved in these effects? There are two dominant explanations in the literature, the first is that with low diversity, the probability of having species with the key traits present in the community is reduced and so productivity falls (the sampling effect). An alternative explanation is that at diversity falls, so fewer species utilize resources less completely (niche complementarity). Both of these hypotheses are focused on resource acquisition and use but nearly 50 years ago, Elton (1958) proposed that reduced biodiversity would increase the severity of diseases. An important hypothesis and one very relevant to the objective of this book. Does parasitism increase in severity with reduced biodiversity? Can the influence of disease be an important mechanism that reduces plant productivity? Not trivial questions but questions that help us identify the role of parasites in the ecosystem. Two recent studies have shown us that that biodiversity really does influence disease severity. The first examines the pathogen load of grass swards and shows that biodiversity does indeed reduce disease severity and influences productivity from an ecosystem. The second examines the tick borne zoonotic Lyme disease and shows that hat reduced wildlife biodiversity increases the risk of infection to humans.

1.3.1 Fungal pathogens and plant biodiversity

Foliar fungal pathogen spores are spread by wind and rain and are common amongst grass species; they darken leaves, reduce leaf life span through the loss of nutrients and photosynthate and so depress productivity. Mitchell (2003) demonstrated experimentally

that excluding these pathogens from intact grassland with fungicide dramatically increased root production and biomass by increasing leaf longevity and photosynthetic capability. The interesting aspect of this work was that he also undertook a factorial experiment that included insect herbivores along with the pathogens and showed that herbivory had relatively little impact compared with pathogens and thus demonstrated that pathogens were potentially the important regulators of ecosystem processes.

The hypothesis that the severity of disease is greater when diversity is reduced is explained in plants because reduced biodiversity results in increased abundance and in turn this facilitates specialist pathogen transmission. This is well illustrated by studies in agriculture where the impact of a pathogen in a cereal system is reduced when mixtures of multiple genotypes of one crop species are grown together (e.g. Zhu et al 2000). In another excellent study Mitchell et al (2002) tested the diversity-disease hypothesis by using experimental communities of perennial grassland plants where diversity was controlled directly by hand weeding. They showed that decreased diversity increased pathogen load to an extent that pathogen load was three times greater in a monoculture than in a plot with 24 grass species, the equivalent to a natural system (Figure 2). They showed that increased pathogen load and the severity of the attack was essentially the consequence of changes in host abundance although they also noted that changes in the relative species composition played an important role.

These studies are interesting and important since they show us that as species diversity increases so the impact of the pathogens falls and plant productivity increases. At the current time it is not clear how much these pathogens account for the total changes in productivity observed and as Mitchell points out the overall impact depends on species composition and the presence of basal species. Nevertheless one point is clear, parasites are an important component that has a dominating influence on ecosystem functioning and their presence should not be ignored.

1.3.2 Lyme disease and biodiversity

The previous section examined how pathogen load changed with the diversity of grassland species and showed that as diversity fell, so relative abundance of the few remaining species increased and this resulted in increased transmission of the specialist pathogens. However one of the interesting findings was that while the overall pattern was clear, there was high variation between trials since the species composition varied and certain species influenced the pathogen load more than others. When species richness falls, the order of loss is often not random, we can predict that some species are more vulnerable to disturbance than others and these will be lost first. If these species vulnerable to disturbance are also less important in disease transmission then this will result in a relative increase in the pathogen load over and above changes in relative density.

What happens when the less competent hosts are lost first? LoGiudice et al. (2002) examined this question in the zoonotic Lyme disease system of North America, a disease caused by the spirochete, *Borrelia burgdorferi* that is transmitted between hosts by the tick, *Ixodes scapularis*. The immature stages of the ticks are generalists and feed on a wide range of mammalian, avian and reptilian hosts but while these hosts may provide a blood meal for the ticks they are not all competent hosts for the transmission of the Lyme disease pathogen. In other words, when an infected tick bites some hosts the pathogen is introduced into a “dead end” host and lost from the system so that when naïve ticks bite the host they do not become infected. The dominant competent host in the system is the white footed mouse, they can infect up to 90% of larval ticks depending on how many of the larvae are feeding on the non competent hosts. When woodland habitat is degraded from pristine woodland to wood lots, the charismatic larger species (and those not competent for Lyme disease transmission) are often the first to go while the mice are invariably the last species. As species richness falls so proportionately more ticks feed on the white footed mouse and since the mouse is a competent hosts, and many of the other hosts are not competent, the overall level of prevalence in the ticks should increase and the risk of infection to humans increase. LoGiudice et al (2003) tested this hypothesis by showing that the non-mouse hosts are relatively poor reservoirs for Lyme disease and dilute the disease by feeding ticks but not infecting them with the spirochete. They

captured the 10 main groups of hosts, estimated their relative density, counted the immature stages of ticks on each, the proportion that engorged and then molted and also estimated the relative abundance of the host to transmit the pathogen. These unique data provided a community level insight into the relative role of each vertebrate host species in the transmission of the disease. Since they also knew the approximate order of species loss (mice are lost last) from woodland areas as they become degraded they were able to show clearly that as species diversity increases so the infection prevalence in the nymphs, and so the risk of human infection falls. The mouse is so dominant that the effects depend strongly on what the mouse density is within the study area. Even so, Squirrels had the highest dilution effect reducing prevalence by about 58% whereas shrews provided a rescue effect; they acted to dilute the effects of the most competent mouse hosts but could also maintain the spirochete in the community when mouse density was low.

This study shows that the buffering of disease prevalence is an interesting and important function provided by high biodiversity. The finding is important since it shows us that while the presence of the ticks is important, we must consider the biological role of the different host species. While changes in climate may influence the development and survival of vectors when they are not on the host it may also influence the distribution and abundance of host species that may have a large effect on disease prevalence and risk of infection to humans.

1.4 How does parasitism affect biodiversity? Parasite mediated competition

I now want to turn the biodiversity and parasitism question on its head and ask the inverse of the previous question: how does parasitism affect biodiversity. There are well defined hypotheses that a major driving force behind the evolution of species diversity is parasitism (e.g. Janzen 1970, Connell 1978). Rather than consider this in detail I wanted to introduce an important threat of parasitism to biodiversity and conservation: the effects of a reservoir host on the abundance and existence of a more vulnerable species through the process of apparent competition, sometimes referred to as parasite mediated competition (Price 19xx, Hudson & Greenman 199x). The evidence that parasites may

drive some populations to extinction is frail. There is the clear example of a microsporidian parasite killing the last known individual snail *Partula turgida* (Cunningham & Daszak 1998) but there are few documented cases where parasites alone have driven a species to extinction.

General theory assumes, logically, that the transmission of most parasites can be considered density dependent so if a virulent pathogen is introduced into a susceptible host population it will reduce density but once density is reduced, transmission will fall and the population will not be extirpated. However parasites could lead to local extinction when transmission is frequency dependent, independent of density but dependent on the contact rate between conspecifics. So for example when wild dog density fell in the Serengeti, they still lived in packs and held their social structure with daily contact rates and the reduced overall density did not mean the few remaining individuals were spread independent of each other. Sexually transmitted diseases depend on the frequency of partner exchange and not host density so HIV or syphilis increases with the number of partners each infected individual has a sexual relationship with and not the total density of hosts in the population. HIV would fade out in a dense community of strictly monogamous couples unless maintained through other forms of transmission such as blood transfusion. Similarly vector borne disease are frequency dependent since transmission depends on being bitten by a vector then the more often a host is bitten the more likely they are of being infected, irrespective of host density. Indeed vector borne diseases often exhibit inverse density dependence since as host density decreases, due to parasite induced mortality, so the remaining vectors focus on a smaller and smaller number of hosts thus increasing the likelihood of them being exposed and dying from the vector borne disease. In this instance disease could drive species to extinction.

Another way in which parasites can reduce biodiversity is in shared parasitism where two or more species share a parasite, in one the parasite causes little mortality but this species sustains the infection and there is between-species transmission such that a second, more vulnerable species receives the infection and suffers significant mortality and eventually becomes wiped out. This is parasite mediated competition. A preliminary glance at the

literature and some of the reviews indicates that parasite mediated competition may indeed be rife. However few workers have examined the effects of parasites in detail and clearly separated the effects of direct from parasite mediated competition in the wild. One of the classic experiments sometimes referred to as parasite mediated competition is the laboratory study of *Tribolium* beetles by Park (1948) where he showed that a competitive interaction between two species was reversed when an *Adelina* parasite was added to the system but this appears to be a special case where the competitive ability of one species is reduced by the parasite rather than indirect competition via a shared parasite. The clearest example is the beautifully designed laboratory study by Bonsall & Hassell (1997) summarized in Chapter 8, but here I wish to highlight two field studies that have examined parasite mediated competition in the wild.

1.4.1 Squirrel invasion and parapox virus

Since its first introduction into Britain, the grey squirrel has spread and replaced the red squirrel. While the dominant theory was resource competition was the underlying cause of the replacement, simulation modeling indicates that this alone can not account for the rate and pattern of red squirrel decline (Rushton et al 1997). When the grey squirrel was introduced, it brought with it a parapox virus that may have a big impact on red squirrels but not grey squirrels and may have had at least a helping hand in the demise of the red squirrel. A critical piece of evidence comes from the study by Tompkins et al (2002) who showed that the virus caused a severe and deleterious disease in the red squirrels but had very little effect on the grey squirrels. Detailed modeling of the system shows that the parapox virus was likely to have played a critical role in the demise of the red squirrel even though the prevalence of infection was low and may have led to previous workers dismissing its role (Tompkins et al 200x). These studies show that parasite mediated competition could be taking place but at this stage it is not clear whether this acts alone or the pathogen may be interfering with aspects of competitive ability. Disentangling the effects of parasites on direct competition from parasite mediated competition is not simple but the introduced squirrel appears to be having an effect on the endemic species through the effects of parasitism.

1.4.2 Game birds and gastrointestinal nematodes

One of the clearest examples of parasite mediated competition are some detailed studies on the shared nematode parasite *Heterakis gallinarum* that infects both the ring necked pheasant and grey partridges (Tompkins et al JAE, Parasitology). In this system it would appear that there is little direct competition between the two hosts but the impact of the parasite on the grey partridge is enough to drive it to localized extinction. Intensification of agriculture in the British countryside has led to the loss of weeds and the invertebrates associated with them leading to the demise of the grey partridge (Potts 198x). Land owners and farmers faced with the loss of a quarry species replaced the partridge by rearing and releasing large numbers of pheasants. A number of farms and other areas have extensified their land management practices, introduced conservation headlands, beetle banks and encouraged the habitat so partridges should recover. Unfortunately many of these have failed and there appears to be some other process acting that could be preventing recovery and this maybe parasite mediated competition.

Tompkins et al () infected pheasants and partridges in captivity with 500 eggs of the gastrointestinal nematode *Heterakis gallinarum* and then followed changes in body condition and worm egg production. Both host species exhibited the classic self-cure and after 100 days had cleared infection but the fitness of the worms was roughly 2 orders of magnitude greater in the pheasants in that each worm introduced into the pheasant produced 100 times more eggs than the in the partridge. What was more, the impact of the worms was such that the partridges lost condition while the pheasants did not, in other words worms in pheasants produce more infective stages but only partridges suffer from the infection indicating that the pheasants are potentially an important reservoir of infection. They then released partridges into 5 different areas where they had monitored infections in wild pheasants and the uptake of infective stages in the partridges was directly dependent on the intensities in the pheasants (Tompkins et al). This demonstrated that the partridges were being infected through a common pool of infective stages and the larger this was the more this increased infection in the partridges and the bigger the impact on partridge condition. By modeling the multiple host system they were

then able to demonstrate that these effects were sufficient to lead to the local demise of the partridges by the pheasants through the process of parasite mediated competition (Tompkins et al.). This is one of the few field based studies to show clearly that parasite mediated competition operates in the wild and that parasites have an effect on biodiversity.

1.5 The role of parasites in ecosystems

In the first section of this chapter I looked at how a specialist parasite (*Trichostrongylus tenuis* in red grouse) can influence host dynamics and so other natural enemies, the complexities of a specialist pathogen with a generalist vector (Louping ill) and then the far reaching repercussions of a generalist pathogen in a relatively complex foodweb (Rinderpest in the Serengeti). I think that detailed studies on the impacts and transmission of parasites provides us with an understanding of how parasites influence host dynamics and thus allows us to examine the consequences of parasitism in an ecosystem. These first order interactions are examined in more detail in Chapter 3.

In the second section I touched on two of the central questions to this book: How does biodiversity affect parasitism? and how does parasitism affect biodiversity? I have but scratched the surface and I hope by doing so wetted your appetite for the main meal of the tome that follows. The following chapter (Chapter 1) takes this approach much further by applying our understanding of ecosystem science to the world of parasitism. One thing becomes abundantly clear, parasitism is a dominant part of many ecosystems and while there may be few measures of the relative biomass of parasites in a system we should appreciate that parasites often have a very large turnover rate, so parasites may have a relatively large effect on energy flow. The whole idea that parasites redirect energy away from other trophic levels and how they operate within a spatially heterogeneous environment is explored fully Chapter 4. These discussions are never far from the dominating concerns that many environmentalists, as well as biologists, have about our ecosystems, the loss of biodiversity and what we can do about it from a conservation strategy, as examined in Chapter 8. Perhaps we could take a few lessons

from the few but beautifully detailed studies that have been undertaken in disturbed (Chapter 7) and hostile environments (Chapter 6).

One key issue I was keen to address but felt I should leave for others more qualified is that fact that parasites and hosts are in a wonderful evolutionary tension where the host develops increased resistance and the parasite generates the means of avoiding the host's response. The immune system is a complex and highly adaptive system that must outstrip the within host growth of the pathogen and at the same time carry a memory. This memory can have important implications to the way the infra-community of the parasites develops and is observed (e.g. Lello et al 2004) and current levels of infection within any mature vertebrate are the ghost of infection past. The whole manner in which parasite communities are structured in ecosystems is examined in detail in Chapter 2 and then examined in relation to the structure of the food web in Chapter 4. The evolutionary tension I refer to of course not only influences the host's response but also the response of the pathogen, this includes features such as parasite induced susceptibility to infection (explored in Chapter 9) and the important effects of anthropogenic impacts on the environment, examined in Chapter 10. The integration of evolutionary principles with those of epidemiology and ecosystems is extremely exciting and I feel the need to point you at a recent exciting paper by Grenfell et al (2004) that brings together an evolutionary, phylogenetic approach with our understanding of epidemiology into a new field of investigation that has become known as phylodynamics. I suspect many of the dualities we observe in parasite-host systems arise because of the tensions between the evolutionary and epidemiological characteristics of parasites.

There is little doubt that the whole concept of how parasites fit into the ecosystem, how they interact with the community of hosts and climatic changes is rapidly becoming an exciting new field. One group of workers calls this integrated discipline conservation medicine (Tarbor et al 200x, Hudson 2004) and there is now a new journal of *EcoHealth* which publishes papers on disease and ecosystem sustainability. This journal and study area examines in detail how the emergence and effects of pathogens and parasites interact with anthropogenic pollutants and the environment to influence disease dynamics. The

vertical integration of the subject is essential; at the sub-cellular level we need to understand the very binding processes that allow a virus to enter a cell of a specific host and in the case of zoonotic diseases a second host that is phylogenetically distant (e.g. Hanta virus in mice and humans, West Nile virus in birds, humans and horses). We need to embrace the machinations that results in relatively high rates of mutation in the RNA viruses, the genetic processes of recombination that can lead to the evolution of new strains and how these interact with the complex arms of the hosts immune system. At the cellular level we need to examine how parasites locate the organ they inhabit, the complexities and shifting sands of antigen expression and then the consequences this can have for such subjects as assessing optimal mates for sexual reproduction. At the level of the individual host, we need to include the patterns of the parasite communities within and between hosts and how species interact and can influence the within host selective forces that occur during the course of infection. One major development over the past 25 years has been the production of logical, generic models that integrate the findings at the individual level and then predict the epidemiological consequences at the population level (Anderson & May 1991, Hudson et al 2002). This approach has served to identify the means of controlling infections in a number of important epidemics. We now need to extend these to the community level to understand how and when species may act as reservoirs, the means by which to control them (vaccinate reservoir or target host?) and the ecosystem consequences of our actions. The final frontier is to combine the fundamental aspects of ecology with immunology and evolutionary biology to understand the role of parasites in a dynamic ecosystem.

I want to finish with one final and ironic duality; the perceived importance of parasitism in natural systems is a simple reflection of the importance of disease in the human population. Before the 1950s ecological workers often considered parasitism of major importance in wild host systems. Later, the development of penicillin and vaccination programs reduced the threat of infectious disease to humans and in the same manner the ecological texts of the time played down and usually ignored the role of parasites in population dynamics or community structure. Since then we have seen increased concern over emerging diseases, a series of important epidemics (HIV, SARS, Foot & Mouth,

BSE, Avian Influenza etc.), the development of resistance to anthelmintic drugs and the threats of bio-terrorism all of which have highlighted the role of parasites. In parallel we have seen increased concern and funding for disease related issues and modern ecology texts include parasitism in natural systems in at least one chapter. Modern parasitologists and the ecological parasitologists that are the authors of this book now see that the parasite-host relationship is full of non-linearities that need investigating if we are to control the threats of infectious diseases but at the same time encourages us to appreciate the pivotal role parasites can play in an ecosystem.

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